Mandibular Herpes Zoster

With Report on the Use of Cortisone in a Case with Geniculate Ganglion Symptoms

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• *Herpes zoster, an acute specific viral infection, occurs more commonly than is generally supposed. It should be differentiated from other diseases involving the ear and skin; it must be considered as a possible etiologic agent in some palsies of the facial, glossopharyngeal or vagal nerves.*

The type of cephalic herpes zoster should be carefully differentiated; cases involving the "geniculate zone" may be other than "Ramsay Hunt's syndrome." This syndrome is now defined as a herpes zoster eruption of the external ear at the "geniculate zone" with involvement of the seventh or seventh and eighth nerves.

The "topognostic" method is the best for determining the level at which the facial nerve has been affected.

It is questioned whether there is a single outstanding therapeutic agent for this disease. Cortisone had no apparent therapeutic effect in a case reported herein.

SINCE CEPHALIC HERPES, including Ramsay Hunt's syndrome, is a manifestation of herpes zoster still not completely understood, the present communication deals with the present concept of Hunt's syndrome and some of the overlapping features of this disease and other kinds of cephalic herpes. In a case herein reported upon, cortisone was administered to determine its efficacy.

Herpes zoster is an acute specific virogenic infection usually occurring sporadically, although sometimes in small epidemics. The acute phase lasts a few days to several weeks. The virus is closely related to that of varicella and may be identical. Various factors seem to predispose to this disease such as exposure, fatigue, trauma, debilitation, septic states and (formerly) treatment with heavy metals. One attack confers definite and lasting immunity. Rabbiner[25] stated that it is a comparatively uncommon disease and that a case he reported upon was the first in 43,521 cases of disease of the ear observed on the otolaryngological service of the New York Post-Graduate Hospital. However, it would appear to be more common than stated inasmuch as in many cases the symptoms may not be full-blown and misdiagnosis is a possibility.

The disease is characterized by early symptoms such as fever, malaise and various paresthesias which may include very severe pain in the involved segmental epidermal or mucosal areas. Although it most often affects sensory ganglia and nerves, there may be involvement of motor nerves. If the facial nerve is involved, paralysis may precede or follow the eruption. Within a few days the characteristic lesions, indistinguishable from those of varicella, appear. The spinal fluid contains an increased number of lymphocytes. The course is usually rapid, diminution of the pain and resolution of the epidermal and mucosal lesions occurring within a few days of onset.

Sequelae, although they are rare, may include prolonged neuralgia, noted particularly in aged and debilitated persons, and motor palsy. If the ophthalmic division of the trigeminal nerve is involved, serious damage to the eye may result.

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Herpes zoster should be differentiated from external otitis, mastoiditis, labyrinthitis, erysipelas and diseases which are characterized by vesiculation. It is believed that some cases of idiopathic facial palsy and palsies of the glossopharyngeal and vagal nerves may be aberrant cases of herpes zoster. Certainly before the characteristic lesions occur the diagnosis may not be very apparent.

The present concept of Ramsay Hunt's syndrome includes a zoster eruption of the external ear at the "geniculate zone" associated with involvement of the seventh or seventh and eighth nerves.

Although the disease has been known since early times, Von Baerensprung in 1861 was the first to demonstrate the lesion in the spinal ganglia. Head and Campbell[15] in 1900, in a large series of cases studied at autopsy, noted involvement not only of the posterior root ganglia but also of the posterior nerve roots, the corresponding peripheral nerves and the ascending fibers in the posterior columns of the spinal cord. The chief pathological features were pronounced inflammatory and hemorrhagic

reaction followed by destruction of the neural tissue, resolution and development of fibrous tissue at the site of destruction. Definite affinity for sensory cranial or spinal ganglia and the corresponding neural distribution was noted. Lever[22] stated that the dermal lesions of varicella and herpes zoster, microscopically observed, are very similar. In both there is pronounced degeneration of the epidermal cells producing a "ballooning degeneration" and a unilocular vesicle. He expressed belief that the causative viruses are identical.

Denny-Brown and co-workers,[7] in reporting upon autopsy in a case of occipitocollar and auricular herpes with seventh nerve palsy, noted that the geniculate ganglion was normal and that there was patchy lymphocytic infiltration in the facial nerve. Thus they established that the geniculate ganglion is not the site of the lesion in certain cases of Ramsay Hunt's syndrome.

Hunt[16] in the early part of this century directed much attention to herpes zoster involving the head and neck. He elaborated on the herpetic inflammation of the geniculate ganglion and classified the involvement of this ganglion into four types of increasing severity: (a) herpes oticus, (b) herpes oticus with facial palsy, (c) herpes oticus with facial palsy and hypoacusis, (d) herpes oticus with facial palsy and Meniere's complex.

The distribution of the herpetic eruption involved one or more of the following areas: the facial, auricular or occipitocollar.[17] Hunt believed that the inflammation involved one or more of several ganglia including the gasserian, geniculate, cervical, acoustic, glossopharyngeal and vagal ganglia.[16]

The zoster area for the geniculate ganglion was thought to include the following: "the tympanic membrane, the external auditory canal and meatus, the concha, tragus, anti-tragus, lobe of the ear (external surface), anthelix, and the fossa of the anthelix."[16]

Hunt[18] believed that in cases in which seventh nerve palsy was present with facial or occipitocollar herpes, the geniculate ganglion was involved in the process. Although most of Hunt's conclusions have been accepted, his belief that the geniculate ganglion is involved concomitantly when facial paralysis is associated with a trigeminal or cervical herpes zoster has been invalidated by the evidence obtained at autopsy and subsequent clinical interpretation.

Engstrom and Wohlfart[8] reported four cases of herpes zoster, in two of which there were herpetic lesions and palsy in the distribution of the ninth and tenth nerves, vesicles in the geniculate zone and eighth nerve involvement. The authors believed that the sites of the lesions were limited to the glossopharyngeal and vagal ganglia, although lesions occurred in the geniculate zone and the eighth nerve was involved. They indicated that certain cases of herpes zoster oticus may be of vagoglossopharyngeal origin and questioned whether herpes zoster oticus in the "Hunt's syndrome" is caused by changes in the geniculate ganglion. It was their opinion that Ramsay Hunt's syndrome should include only those cases of herpes oticus with facial palsy and/or lesions of the eighth nerve.

Kidd[20] and other investigators have questioned the existence of a sensory cutaneous function of the seventh nerve. Furlow[12] reported on a case in which the patient had deep pain in the ear initiated by the stimulation of a small area of the external auditory canal. In this patient all conservative measures failed. At operation when the nervus intermedius was touched the pain was duplicated. The nerve was sectioned and there was no further occurrence of the pain. The sensory function of the seventh nerve has been accepted although there is considerable overlapping with the auriculotemporal nerve anteriorly, the second and third cervical nerves posteriorly and the glossopharyngeal and vagal nerves.

Much credit is due Tschiassny for his furtherance of Erb's work in localizing the site of lesions of the facial nerve. Erb[9] divided peripheral palsy into several divisions, the exact site depending on the one or more branches of the nerve involved. He included the following factors: taste, hyperacusis, presence or absence of palsy of the soft palate due to the action of the levator palati, paralysis of the auricular muscles and dryness of the mouth. According to Tschiassny[29, 30] the seventh nerve consists of four portions, two being efferent, namely the motor and secretory portions. The remaining two, sensory and gustatory, are afferent. He expressed belief that the greater superficial pretrosal nerve transmits secretory fibers for the lacrimal gland. Also it was his belief that the fibers transmitting the sensation of taste from the anterior two-thirds of the tongue do not pass via the seventh nerve central to the geniculate ganglion. Thus a lesion of the seventh nerve could be localized to a particular level. Furthermore, he expressed opinion that there are two communications between the seventh and eighth nerves in the internal auditory canal, one communication (termed "supragenicular") going between the eighth and seventh nerves central to the geniculate ganglion; the other ("transgeniculate") between the eighth nerve and the geniculate ganglion. Thus a supragenicular lesion would not involve the function of taste inasmuch as there are no taste fibers present at this level. A transgeniculate lesion would involve all the functions of the seventh nerve including taste and lacrimation.

According to Tschiassny,[31] testing of the lacrimal function by mechanical or chemical irritation of the

nasal mucosa is not a reliable method. However, unilateral absence of lacrimation upon stimulus of an emotional character is much more indicative of unilateral dysfunction.

In mandibular herpes the loss of taste in the homolateral portion of the tongue could be attributed to one or both of the following factors: (1) Severe herpetic glossitis in the distribution of the lingual nerve causing damage to the taste buds in the homolateral portion of the tongue. (2) Herpetic neuritis of the lingual nerve affecting the lingual mucosa and the terminal filaments of the chorda tympani taste fibers, thus involving the chorda tympani nerve indirectly.

There is no unanimity of opinion regarding the central pathways of taste. Ranson,[26] Krieg[21] and Schwartz and Weddell[27] expressed belief that the nervus intermedius transmits the sensation of taste central to the geniculate ganglion from the anterior two-thirds of the tongue. Grinker and Bucy[14] observed that taste fibers within the seventh nerve may possibly enter the petrosal or sphenopalatine ganglion rather than pass through the seventh nerve. Ballenger[2] expressed opinion that lesions central to the geniculate ganglion do not affect the sense of taste. Thus, attempts to place the level of a seventh nerve lesion based on the presence or absence of taste may be on dubious grounds.

Furthermore it is questioned whether the communications between the seventh and eighth nerves in the internal auditory meatus are a definite and constant anatomic fact, considering their filamentous nature. Piersol[24] stated that "these apparent communications between the seventh and eighth nerves are, in fact, only aberrant strands of facial fibers that return to the seventh after temporary association with the acoustic."

At present the best means of localizing the site of a lesion involving the facial nerve is based on the topognostic method elaborated by Tschiassny[29, 30] in which the presence or absence of the functions of the facial nerve are evaluated; however, these may be absent, altered or transient.

THERAPY

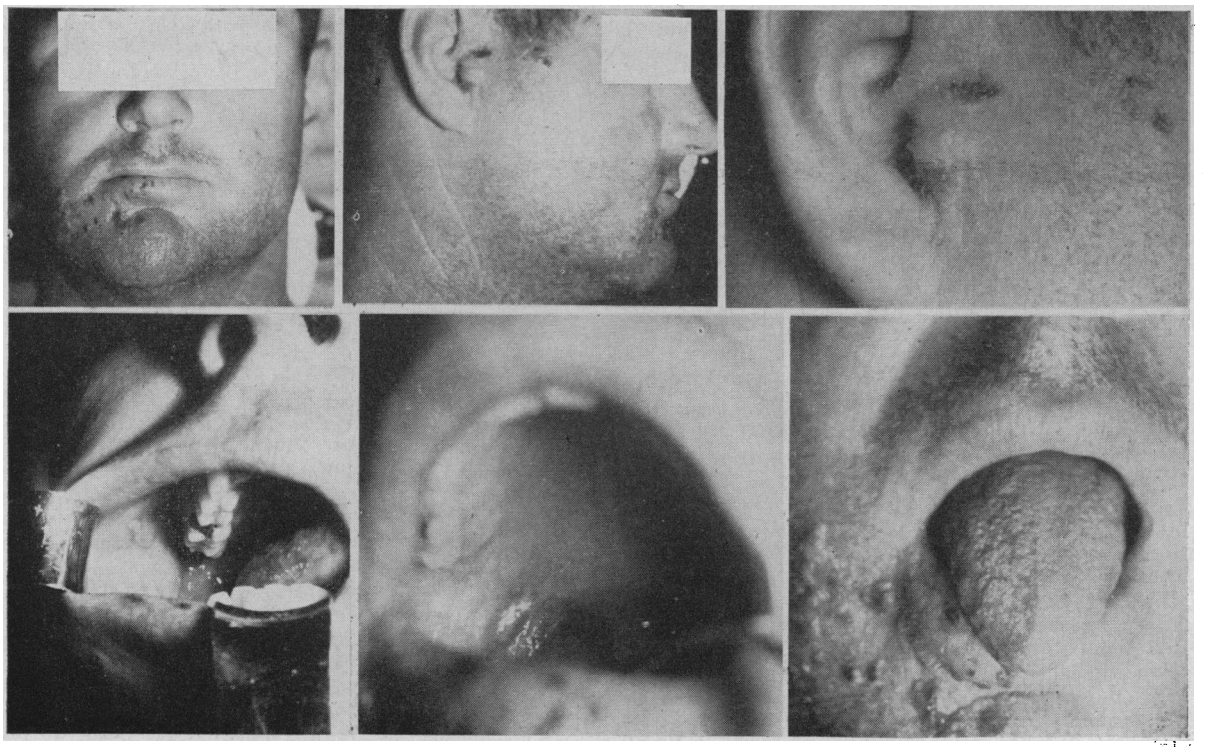
Numerous remedies have been used in the treatment of this disease. Foerster[11] employed roentgenotherapy of the spinal region. He reported that the duration of the zoster eruption was probably shortened and that the pain definitely diminished. Becker[3] believed that good results followed the use of transfusions of blood from convalescent subjects. Dennie, Morgan and Coombs[6] used multiple injections of moccasin venom. In 18 of 20 cases improvement occurred and they believed that this method gave the best results of any single drug that had been used. Marsh[23] considered protamide superior to pituitrin, thiamine chloride, autohemotherapy, sodium iodide or high voltage roentgen therapy. Banthine® appeared to shorten the course of the disease according to Brown, Reekie and Sinclair.[4] Gans[3] employed aureomycin in six cases of herpes zoster and rapid resolution of the disease followed. Shier and Provisor[28] used aureomycin and were impressed by favorable results. Babbage[1] stated that chloramphenicol aided the resolution of the disease. Carter[5] divided a group of 44 consecutive patients with herpes zoster into four groups. The patients in one group were not treated and those in the other three were treated with chloramphenicol and different doses of aureomycin, respectively. Except for the control of secondary infection, the results were essentially the same in all groups. Kass, Aycock and Finland[19] in using aureomycin and chloramphenicol in a series of 72 patients with herpes zoster corroborated Carter's results and concluded that these two agents were not particularly efficacious. Fishman,[10] reporting on the use of a daily maintenance dose of 65 mg. of cortisone for a patient with rheumatoid arthritis, in whom herpetic lesions appeared on the right shoulder during the eighth month of therapy, concluded that cortisone neither affected the course of herpes zoster nor alleviated the pain associated with it. There are few reports in the literature regarding the use of cortisone. Additional remedies proposed for zoster are very numerous and include pitressin, thiamine, diphtheria antitoxin, liver extract, Etamon,® ergot preparations, dihydroergotamine-45, autohemotherapy and procaine injections into the intervertebral and prevertebral ganglia.

Although herpes zoster is not a fatal disease and sequelae are rare, sometimes the epidermal and mucosal lesions are pronounced and the pain may be very severe. Also, in the case of ophthalmic herpes, the consequences may be serious. Hence it is still necessary to find an effective means of treatment. In the following case cortisone therapy was given.

REPORT OF A CASE

A male patient 38 years of age when first observed had a history of infection involving the right ear beginning approximately three days previously and characterized by pain but no discharge or impairment of hearing. At the onset of the illness he noticed, when combing his hair, sensitivity of the scalp on the right side, and while shaving he noted another sensitive area on the chin. Later there was a more definite stabbing pain deep within the ear. Blisters began to form on the lower lip and chin and there was discomfort about the right eye. Later there was a sensation of tingling and prickling of the entire right side of the face.

When first observed, the patient had small ulcers scattered over the buccal mucosa. There were some vesicles on the lower lip and chin and several just anterior to the root of the helix. The auditory canal appeared approximately normal although the membrana tympani had a bluish tinge.



Involvement of sensory branches of the mandibular nerve.

An audiogram revealed normal hearing. On July 10 there was hypogeusia to sugar, salt, vinegar and quinine and diminished lacrimation as tested by nasal inhalation of onion and formalin. Hyperacusis was absent. Meanwhile the pain had become worse and there were more lesions on the skin and on the buccal mucosa. The vessels of the external auditory canal were engorged and vesiculation was beginning. Parenteral administration of 200 mg. of cortisone daily, given in two equal doses with an eight-hour interval, was begun on July 10 and continued for the next six days. On July 11 the pain was almost unbearable, recurred at short intervals, and did not respond to sedatives and analgesics. The right canal showed considerable ulceration and edema in the "geniculate zone" although the membrana tympani appeared normal. By this time the herpetic lesions were noted to have delineated the entire sensory distribution of the mandibular nerve including the temporal, mandibular, buccal, gingival and lingual areas and the anterior pillar. On July 12 the pain had diminished considerably although no change was noted in the lesions. Within the next several days, however, they began to heal. By July 30 the skin was almost normal although there remained areas of paresthesia in the scalp and about the ear.

COMMENT

In the current concept, Ramsay Hunt's syndrome is limited to a herpetic eruption in the "geniculate zone" with involvement of the seventh or seventh and eighth nerves.

In the case here reported, all functions of the facial nerve except motor function were apparently affected. The "geniculate zone" was included in the site of the eruption. This was not a true "geniculate" eruption and could be attributed to the contiguity of the nerve supply to this area inasmuch as the posterior wall of the external auditory canal is not considered to be supplied by the auriculotemporal nerve.

The loss of the sense of taste would be presumed to be owing to chorda tympani involvement. However, the chorda tympani *per se* was not involved in the herpetic process and the loss of taste was owing to glossitis in the area supplied by the lingual nerve or neuritis of the lingual nerve involving the lingual mucosa and the chorda tympani nerve indirectly.

There is no physiologic explanation for diminished lacrimation in the absence of involvement of the seventh nerve; a temporary reduction is a transient relative finding and is noted by comparison with the normal eye. The discomfort and general status of the patient undoubtedly entered as factors. Furthermore, it is obvious that mandibular herpes would create more symptoms manifesting seventh nerve involvement than would involvement of either of the other two divisions of the trigeminal nerve.

Since the vagus and glossopharyngeal contribute to the nerve supply of the ear, aural symptoms and lesions may be anticipated when either or both of these nerves are involved in herpes zoster.

The best means of determining the site of the lesions is the "topognostic" method based on the level at which the involved branches leave the facial

nerve. The opportunities for postmortem examination to determine the site are few, for herpes zoster is not of itself a fatal disease.

In the case here reported, the pain increased and the epidermal lesions extended for two days following the beginning of cortisone therapy, although it must be noted that the hormone was not given in maximal dosage. Since the duration of herpes zoster is variable, no positive conclusions can be drawn as to the efficacy of cortisone therapy.

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